PULMONARY FUNCTION TESTS IN ISOLATED RHEUMATIC MITRAL STENOSIS

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CERTIFICATE

This is to certify that this dissertation entitled "**PULMONARY FUNCTION TESTS IN ISOLATED RHEUMATIC MITRAL STENOSIS**" submitted by **Dr. N. PERIYASAMY** to The Tamil Nadu Dr.M.G.R. Medical University, Chennai is in partial fulfillment of the requirement for the award of M.D. degree Branch I (General Medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

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DECLARATION

I, **Dr. N. PERIYASAMY** declare that I carried out this work on **"PULMONARY FUNCTION TESTS IN ISOLATED RHEUMATIC MITRAL STENOSIS"** at Department of General Medicine, Government Rajaji Hospital during the period of March 2005 – April 2006. I also declare this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any university, board either in India or abroad.

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the M.D. in General Medicine Degree examination.

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CONTENTS

INTRODUCTION

Rheumatic heart disease is the most commonest acquired heart disease all over the world.

Mitral Stenosis is almost always rheumatic in origin. Two third of the patients with mitral stenosis are females and mixed mitral stenosis and mitral regurgitation are generally rheumatic in origin; Very rarely, MS is congenital. Predominant Mitral stenosis occurs in approximately 40% of patients with rheumatic heart disease. It remains a major problem in developing nations, especially in tropical and semitropical countries.

In Rheumatic stenosis the valve leaflets are diffusely Thickened by fibrous tissue and/or calcific deposits. The mitral commissures fuse, the chordae tendinae fuse and shorten, the valve cusps become rigid and these changes in turn, lead to narrowing at the apex of the valve Calcification of the stenotic mitral valve immobilizes the leaflets and narrows the orifice further. In normal adults the mitral valve orifice is 4-6 $cm^{2.}$ In the presence of significant, i.c., when the orifice is less than approximately $2cm^2$, blood can flow from the to the left ventricle only if propelled by an abnormally elevated left atrioventricular pressure gradient, the hemodynamic hall mark of mitral stenosis. When the mitral valve opening is reduced to $1cm^2$, often referred to as critical mitral stenosis, A left Atrial pressure of approximately 25 mm Hg is required to

maintain a normal cardiac output. The elevated pulmonary venous and pulmonary arterial wedge pressures reduce pulmonary compliance, contributing to exertional dyspnea.

Pulmonary Hypertension

The clinical and haemodynamic features of mitral stenosis are influenced importantly by the level of the pulmonary arterial pressure. Pulmonary hypertension results from

1. Passive backward transmission of the elevated LA Pressure;
2. Pulmonary venous hypertension (reactive pulmonary hypertension)
3. interstial odema in the walls of the small pulmonary vessels; and
4. Organic obliterative changes in the pulmonary vascular bed.

Other pulmonary changes

Fibrous thickening of the walls of the alveoli and pulmonary capillaries occur commonly in mitral stenosis.

Pulmonary function abnormalities, like vital capacity, Total lung capacity, maximum breathing capacity, and oxygen uptake per unit of ventilation are reduced. Airway resistance is abnormally increased and diffusion capacity is also reduced. These changes in the lungs are due, to increased transudation of fluid from the pulmonary capillaries into the interstitial and alveolar spaces. However, the increased capacity of the pulmonary lymphatic system to drain excess fluid retards the development of alveolar edema.

AIMS & OBJECTIVES OF THE STUDY

1. To know the type of pulmonary dysfunction in rheumatic mitral stenosis.
2. Relation between grading of dyspnoea (NYHA Class I to IV) and severity of pulmonary dysfunction.
3. Relation between mitral valvular size (orifice size) and severity of pulmonary dysfunction.
4. Relation between mitral valvular orifice size and grading of dyspnoea.

REVIEW OF LITERATURE

Rheumatic Fever

Acute Rheumatic fever, a non infectious delayed complication of streptococcal sore throat due to group A β hemolytic streptococcus. 0.3% of patients only develop rheumatic fever after a streptococcal throat.

Epidemiology of Rheumatic Fever and heart disease.

Rheumatic Fever is a major cardiovascular health problem in many nations of the world.

The incidence and prevalence of Rheumatic Fever world wide are enormous. The prevalence of Rheumatic Fever and RHD is highly variable, with the highest rates in the middle eastern and sub-saharan African regions. For instance, it is estimated that 3.2 million patients had rheumatic heart disease in India in 1991 and atleast two thirds of these were young children. The incidence is declining in all over the world because of improving socio economic status and reduced crowding and increase use of penicillin.

Agent, Host and Environmental Factors

Only Group A β haemolytic streptococcus throat infections are strong enough to induce an immunologic response are capable of triggering an episode of Rheumatic fever. Acute Rheumatic Fever usually affects children (being most common between 5 and 15) or young adults. The environmental factors like crowding, poverty, health education and

access to medical cave continue to influence the development of Rheumatic Fever.

Social and Economic burden

India has good epidemilogic data on Rheumatic fever. The prevalence of Rheumatic fever or Rheumatic heart disease among school children is 2 to 11 per 1000, with a mean of 6 per 1000.

Indian Government allocates a mere 2% of budget for health related activities, compared with 5 to 10% in the developed world.

Pathogenesis

Rheumatic fever is a immune (cellular and antibody) mediated injury. The current understanding proposes that rheumatogenic streptococci contain multiple antigenic determinants that mimic normal human tissue antigens; these antigens are recognized as Foreign by the susceptible host and result in a hyperactive immune response (both humoral and cellular)

Group A polysuccharide has an N-acetyl glucosamine moiety that cross reacts with antibodies to the heart valve tissue and patients with significant valvular heart disease have an excess of such cross-reactive antibodies. Antibodies to the streptococcal peptidoglycan complexes have been implicated in rheumatic arthritis. The streptococcal M protein has homology with the cardiac contractile proteins including myosin and

tropomyosin and structural proteins like keratin, laminin and vimentin, which are common in the cardiac interstitium.

Cellular immunity instead may contribute significant T cell infiltration in the valvular tissue and synovium. Membrane antigens of group A streptococci can stimulate T cell cytotoxic for cardiac but not skeletal muscle cell.

Rheumatic Fever risk has been associated with increased prevalence of HLA- DR4 in the United States and Saudi Arabia and with increased prevalence of DR3 and DQW2 in India. However, DR2 has increased prevalence in the African American population in the united states, whereas it seems to be protective in Indians.

Pathologic characteristics of cardiac involvement

RF is considered a multisystem connective tissue disease and is characterized by inflammatory, either exudative or Fibrotic, lesions in a number of systems, including the heart, joints and subcutaneous tissue. The Aschoff granuloma is the pathologic hall mark of Rheumatic fever. It consists of a central area of fibrinoid necrosis surrounded ‘by cells of histiocytic – macrophagic origin that show a typical “Owl eye shaped” nucleus. These cells are usually found in the sub endocardial or perivascular regions, in the myocardium and sometimes in the pericardium

Macroscopic appearance is fibrinous pericarditis with Epicardial involvement:

Pinhead vegetations on valve leaflet tissue; microscopic appearance is Fibronoid necrosis in the valve leaflet, cellular infiltrates, and neovascularization of valve. Macroscopically valves are dull and thickened, unlike the smooth pliable normal appearance, and show small verrucous vegetations on the atrial surface of the mitral valve, the chords and the ventricular surface of the aortic valve. The valves are significantly inflamed and edematous and demonstrate extensive pallisading mononuclear infiltration. Aschoff bodies are occasionally seen. There is granulation tissue and fibrous scarring in the late stages of the disease. Aschoff bodies are seen in 30% to 40% of patients with proven or suspected carditis.

Clinical Features

Joint symptoms

Arthritis is a major manifestation of Rheumatic Fever. Arthritis occurs in more than 2/3rd of patients. Large joints of the extremity are usually involved, although smaller joints including those in the hand and feet may occasionally be inflamed. Hip and spine or axial joints are rarely involved. The joints are inflamed at various intervals, each cycle lasts several days, and this gives a migratory character to joint pains

arthritis resolves in most patients within 3 to 4 weeks and does not result in any permanent damage.

Carditis

Rheumatic carditis is an early manifestation; almost 80% of patient who develop carditis do so within the first 2 weeks of the onset of Rheumatic Fever. The most common evidence of carditis is a blowing pansystolic Mitral regurgitation murmur. It is usually grade II to IV, radiates to the axilla, and sometimes is associated with apical, low pitched, short, middiastolic rumble without a presystolic accentuation (carey coombs murmur). Valvular stenosis does not occur in Acute Rheumatic carditis. Mitral regurgitation is due to Active inflammatory valvulitis, valve prolapse, annular dysfunction or dilatation and ventricular enlargement. The aschoff nodules have a predilection for valve rings and this may account for annular dysfunction and dilatation.

Myocarditis is often indicated by the presence of a soft first sound, gallop sounds, cardiomegaly and / or congestive cardiac failure. Myocardial biopsies in patient with active rheumatic carditis do not show significant evidence of myocyte damage. It is now generally believed that congestive heart failure in patients with acute rheumatic carditis is caused by severe mitral regurgitation.

Clinical rheumatic pericarditis occurs in 6 to 15% of patient during the acute stage of rheumatic Fever and the presence of a pericardial

friction rub in this setting is evidence for rheumatic carditis. Presence of pericarditis indicates severe carditis.

Neurologic manifestation

Chorea is usually a late manifestation and appears several weeks after an acute attack of Rheumatic fever at a time when other manifestation have disappeared and patients often do not fulfill the Jones criteria. The onset is usually gradual. The patient appears increasingly nervous, is dysarthric, makes grimacing gestures, had difficulty in writing and shows characteristic purposeless movements of the arms and legs. These involuntary movements are increased by effort or excitement, are absent during sleep, and may be associated with muscular weakness. The pathogenesis of chorea remains unclear. Sydenham's chorea is usually a self limited condition and recovers without residua.

Skin manifestations

Subcutaneous nodules, a late manifestation of Rheumatic fever, occur in 1% to 21% of patients and the presence of subcutaneous nodules usually suggests underlying carditis. The nodules are firm and painless, 0.5 to 3 cm and are usually on bony proninance or vertebral spinous processes and on extensor tendons. They usually appear in crops and disappear within 8 to 12 weeks in most patients.

Similar to subncutaneous nodules, erythema marginatum is usually indicative of underlying carditis. Erythema marginatum, unlike

subcutaneous nodules, however can be an early or a late manifestation and can be present in the absence of features to indicate active Rheumatic fever. It occurs in less than 10% to 15 % of patients, is present on the trunk and proximal extremities as a serpigenous macular non pruritic rash, and is often very evanescent.

Other features

A number of other signs and symptoms seen in patients with acute rheumatic fever and in view of their nonspecific nature are called minor manifestation for diagnostic purposes. These include fever, prolonged PR interval, and elevated acute-phase reactants.

The Jone's criteria for diagnosis of acute Rheumatic fever

Major criteria	**Minor criteria**
Carditis	Previous Rheumatic Fever or RHD
Polyarthtis	Arthralgia
Chorea	Fever
Subcutaneous nodules	ESR
Erythema marginatum	Positive Creative Protein
	Leukocytosis
	Prolonged PR interval

Needs two major criteria or one major plus two minor criteria for diagnosis. Needs supportive evidence for recent streptococcal infection for all diagnosis. The ASO titre should be more than 240 units in adults

and more than 330 units in children. The best diagnostic specificity is obtained by demonstrating an interval increase in ASO in two paired serial samples. Increased ASO titres are seen in within 7 to 10 days. Anti DNase B which remain elevated for several months after even uncomplicated streptococcal infections.

Role for Echocardiography

Clinically detectable valvular regurgitation (Usually mitral and occasionally Aortic) is the hallmark of Acute carditis. Echo cardiography consistently demonstrate valve regurgitation not detectable with clinical examination.

Investigations in Acute rheumatic Fever

1. Evidence of a systemic illness (Non specific) Leukocytosis, raised ESR, raised CRP.
2. Evidence of proceeding streptococcal infection (specific) Throat Swab culture: GroupA β–haemolytic streptococci Antistreptolysin O antibodies (ASO titres)
3. Evidence of carditis

Chest radiograph : Cardiomegaly; pulmonary congestion

ECG : first and second degree heart block; features of pericarditis; T wave inversion; reduction in QRS voltages

Echocardiography : Cardiac dilatation and valve abnormalities.

Chronic rheumatic heart disease

Chromic valvular heart disease develops in at least half of those affected by rheumatic fever with carditis. Two-thirds of cases occur in women. Some episodes of rheumatic Fever may pass unrecognized and it is only possible to elicit a history of Rheumatic Fever or chorea in about half of all patients with chronic rheumatic heart disease. The mitral valve is affected in more than 90% of cases; The aortic is the next most frequently affected valve, followed by tricuspid and then the pulmonary valve. Isolated mitral stenosis accounts for about 25% of all cases of rheumatic disease and an additional 40% have mixed mitral stenosis and regurgitation.

Pathology

In contrast to destructive lytic process of acute rheumatic fever, the main pathological process in chronic rheumatic heart disease is progressive Fibrosis. The heart valves are predominantly affected but involvement of pericardium and myocardian may contribute to heart failure and conduction disorders. Fusion of mitral valve commissures and shortening of the chordae tendineae may lead to mitral stenosis with or without regurgitation. Similar changes in the aortic and tricuspid valve produce distortion and rigidity of the cusps, leading to stenosis and or regurgitation.

Pathophysiology

In normal adults the mitral valve orifice is 4-6 cm^2. in the presence of significant obstruction, I.e when the orifice is less than approximately $2cm^2$, blood flow from the LA to the left ventricle (LV) only if propelled by an abnormally elevated left atrio-ventricular pressure gradient, the hemodynamic hall mark of mitral stenosis. When the mitral valve opening is reduced to $1cm^2$, often referred to as "critical" MS, Left Atrial pressure of approximately 25 mm Hg is required to maintain a normal cardiac output. The elevated pulmonary venous and pulmonary arterial PA wedge pressure reduce pulmonary compliance, contributing to exert ional dyspnea.

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Symptoms of mitral stenosis

1. Breathlessness
2. Fatigue
3. Oedema, ascites
4. Palpitation
5. Haemoptysis
6. Cough with expectations
7. Chest pain
8. Symptoms of Thrombo embolic complications e.g. Stroke, Ischaemic limb)

Signs of Mitral Stenosis

1. Atrial fibrillation
2. Mitral facies
3. Auscultation : Loud first heart sound, opening snap, middiastolic murmur
4. Signs of raised pulmonary capillary pressure, crepitations, pulmonary oedema, effusions.
5. Signs of pulmonary hypertension, RV heave, loud P_2

Investigations in mitral stenosis

ECG

Lt atrial hypertrophy

Right ventricular hypertrophy

Chest radiograph

Enlarged left atium

Signs of pulmonary venous congestion

Echo

Thickened immobile cusps

Reduced valve area

Reduced rate of diastolic filling of LV. According to mitral valve orifice area severity of mitral stenosis is graded into

1. Mild 1.5 - 2.0 cm^2
2. Moderate 1.0 – 1.5 cm^2
3. Severe <1 cm^2

Doppler

Pressure gradient across mitral valve

Pulmonary artery Pressure.

Cardiac cathetorisation

Pressure gradient between LA (or pulmonary wedge) and LV

Pulmonary function abnormalities

Pulmonary function abnormalities denote a disease of either airways or lung parenchyma or both interfering with normal alveolo-arterial gas exchange.

Broadly pulmonary function abnormalities are categorized into 2 patterns :-

1. Obstructive
2. Restrictive

Obstructive pattern of abnormality is classically observed in cases of chronic obstructive pulmonary disease (COPD) whereas Interstitial lung diseases are the prototype for restrictive pattern of abnormality.

Patterns of abnormal ventilatory capacity

	FEV_1	FVC	FEV_1/FVC
Obstructive	↓↓	↓/ N	↓
Restrictive	↓	↓↓	↑/ N

This segregation into either of the pattern is done based on analysis of pulmonary function tests which encompasses a multitude of indicators as described below:

Pulmonary Function Tests

Even though many tests are present to know the lung dysfunction, PFT is easiest and one of the best tests to know ventilation dysfunction.

Definitions

Tital volume – the amount of air moves into lungs with each inspiration (or exhale in each expiration).

Inspiratory reserve volume

The air inspired with a maximal inspiratory effort in excess of the tidal volume.

Expiratory Reserve Volume

The volume expelled by an active expiratory effort after passive expiration

Residual volume

The Air left in the lungs after a maximal expiratory effort.

Vital Capacity

The largest amount of air that can be exhaled after a maximal inspiratory effort.

The fraction of vital capacity expired during the first second divided by the vital capacity is FEV_1.

Spirometry

Introduction

Spirometry is a test of lung function that measures how much and how quickly air can be moved into and out of the lungs. The measurements are made using a spirometer.

A spirometer is an instrument used to measure respired volumes and flows (i.e. spirometry). Many spirometers are able to measure both inspiratory and expiratory airflow.

How to perform spirometry

Spirometry requires maximal effort from the patient and it takes time to perform quality spirometry. It is essential the procedure is carefully and clearly explained and to actively coax and motivate the patient to perform maximally. The volume and flow parameters measured are defined in terms of maximal effort and maximal exhaled volume. The performance of spirometry while seated upright in a chair is

preferable to standing as this is the most stable position should the patient experience dizziness during the test. The seated position is also preferable for patient with urinary incontinence who may otherwise limit the expiratory effort.

The key steps are to urge the patient to:

- Breathe in fully (the lungs must be absolutely full).
- Seal the lips around the mouthpiece and immediately.
- Blast the air out as fast and as far as possible until the lungs are completely empty.
- Repeat the test until three acceptable and reproducible results are obtained (up to a maximum of 8 efforts)
- The highest FEV_1 and FVC should be reported, even if they come from separate blows.

Contraindications

Spirometry is a very safe procedure. However, it is physically demanding as it requires maximal patient effort and it involves the generation of high airway and intrathoracic pressures. It is advisable that spirometry be delayed / abandoned for:

- Recent eye surgery.
- Recent thoracic and abdominal surgery.
- Aneurysms (e.g. cerebral, abdominal).

- ❖ Unstable cardiac function.
- ❖ Haemoptysis of unknown cause (e.g.? TB)
- ❖ Pneumothorax.
- ❖ Chest and abdominal pain.
- ❖ Nausea and diarrhoea.
- ❖ Inability to comprehend the instructions.

This a simple method for studying the pulmonary ventilation by recording the volume movement of air into and out of the lungs. In this test, the subject inhales maximally to total lung capacity (TLC) and then exhales as rapidly and forcefully as possible into the turbine of the spirometer, which calculates the flow rates and volume measurements. The flow rates can calculated from the 'spirogram' which is a plot of volume verses time, and the volume can be calculated from the 'flow-volume tracings' which is a plot of airflow verses the expired or inspired lung volume.

Flow rates

Forced Vital Capacity (FVC) is the maximal volume of gas which can be expired from the lungs during a forced expiration from a position of full inspiration. The FVC can be subdivided into the Forced Expiratory Volume in the first second (FEV_1), represents the integrated flow over the first second of expiration and reflects airway narrowing during expiration.

It is effort independent and normally 70% - 80% of the FVC is expired in the first second.

A more sensitive means of evaluating airway obstruction is therefore, to express the forced expired volume as a percentage of vital capacity, abbreviated as FEV_1% (or FEV_1/FVC). This ratio is relatively independent of the patient's size, and is a specific measure of airway obstruction with or without associated restriction of lung volumes. Normally it is 75% or greater.

Another way of assessing airflow obstruction is to measure specific flow rates. Peak Expiratory Flow Rate (PEFR) is a defined as the maximum flow achievable from a forced expiration starting at full inspiration with an open glottis. It measures the maximum expiratory flow rate over the first 10 millisecond. PEFR is reduced by larger airway narrowing due to asthma, COPD, vocal cord palsy and expiratory muscle weakness etc. The Forced Expiratory Flow from 200 ml to 1200 ml below maximal inspiration (FEF_{2-12}) can also be measured. FEF_{2-12}, also called Maximal Expiratory Flow Rate (MEFR) is used to evaluate the portion of the curve most affected by obstruction of large airways and is most responsive to bronchodilators. FEF_{2-12} and PEFR are useful in the diagnosis and response to bronchodilators in asthma and are very much effort-dependent. Flow from 25 to 75 percent of the total FVC, termed $FEF_{25\%-75\%}$ is another measurement which was originally called as

Maximal Mid-expiratory flow Rate (MMFR). This is effort independent and is very sensitive to airflow obstruction in peripheral, small airways, where disease of chronic airflow obstruction are thought to begin. $FEF_{25\%-75\%}$ is dependent on FVC, in other words, it is the average flow rate during the middle two quarters of FVC.

Flow Volumes

Flow volume curve, a recording during spirometry, of the expiratory flow plotted against expired volume, instead of time, resemble a triangular shaped envelope. At the point where 25% of the vital capacity has been exhaled, this flow rate is termed V_{max25} or $FEF_{25\%}$ when 50% of the vital capacity has been exhaled it is termed V_{max50} or $FEF_{50\%}$ and at 75 of vital capacity it is vmax75 or $FEF_{75\%}$ which refers to the maximal expiratory flow when 75% of FVC remain to be exhaled. Similarly $FEF_{50\%}$ and $FEF_{75\%}$ correspond to $MEF_{50\%}$ and $MEF_{25\%}$ respectively.

The inspiratory portion of the curve is helpful in distinguishing large airway obstruction which occurs above the level of thoracic inlet from obstruction which occurs below this level. Large airway obstruction above the thoracic inlet results in a plateau of the flow rate on the inspiratory portion of the curve, while the expiratory portion is affected when the flow limiting portion is within the thoracic cavity.

$FEF_{75\%}$ is though to be very sensitive to detect early small airway obstruction. In early small airway disease, the only abnormality detected may be reduced $FEF_{75\%}$ and $FEF_{50\%}$ with normal PEFR and FEV_1.

It is a well studied fact that patients with rheumatic mitral stenosis are prone to develop pulmonary ventillatory dysfunction and the severity of mitral stenosis influence the degree of lung function impairment.

In an analysis of 105 patientgs with rheumatic mitral valve diseases [20-21% patients with mitral stenosis and 80-84% with combined mitral valve disease with a stenosis predominance] Hryniewiecki T et al. observed that 93.5% patients had documented airway function disturbance. Using a diagnostic criteria as MEF 50<60% of predicted value, they found out that peripheral airway obstruction had a significant correlation to severity of mitral stenosis. Also bronchial hyperreactivity was noted in 23% patients, but main bronchus obstruction was not significantly present in association with any of the analyzed cardiological parameters.

In another study in 60 patients correlating spirometry abnormalities with different grades of severity of mitral stenosis, Chatterji et al., observed that FVC values were reduced in direct proportion to PAP (Pulmonary Artery Pressure), LAP (Left Atrial pressure) and Mitral valve area (MAV). FEV 1% was also found to be uniformly reduced according to the severity of mitral stenosis and they concluded that mitral stenosis

was associated with a moderate restrictive defect with an inverse correlation and there was no significant involvement of larger airways.

Mahmoud. M Nour et al, in an analysis comparing patients with simple mitral stenosis and those with advanced mitral stenosis scheduled for valve replacement surgery (MVR) confirmed that patients with mitral stenosis have both restrictive and obstructive pattern of pulmonary dysfunction which correlated with the severity of mitral stenosis and with respiratory symptoms.

In another study from the same institution they illustrated that airway obstruction in mitral stenosis in partially reversible with salbutamol nebulization in both pre and post MVR operative cases. This implicated that in patients with mitral stenosis with dyspnoea, doing pulmonary function test would assist in sorting and patients who can be provided symptomatic improvement by supplementing salbutamol nebulization for the reversible small airway obstruction.

MATERIALS AND METHODS

This was a case control – cross sectional study conducted at Govt. Rajaji hospital, Madurai. The study population was divided into two groups.

Group I consisted of 60 Isolated Rh. Mitral stenosis, diagnosed by echo cardiography. Group consisted of 20 Non smoking, healthy volunteers, who served as controls.

Group I (N=60) inclusion criteria

1. Age - 10-51
2. Sex - Male or Female
3. Disease - Isolated Rheumatic Mitral stenosis

Exclusion Criteria

1. H/o smoking
2. H/o Alcohol
3. H/o Any major illness or H/o hospitalization during the past 6 months.
4. History, clinical, radiological evidence of respiratory illness.
5. Past H/o Bronchial asthma, pulmonary tuberculosis, COPD.
6. Aortic valve disease, IHD, uncontrolled hypertension or any other congenital heart disease.

Group II (n = 20) inclusion criteria

1. Age 18-59
2. Sex Male or Female
3. Non smoker

Exclusion criteria

1. H/o. smoking
2. H/o. Alcoholism
3. H/o. recent respiratory illness or Recent hospitalization
4. H/o. Cardiovascular disease
5. History, Clinical, radiological evidence of respiratory illness.
6. Past H/o. bronchial Asthma, pulmonary Tuberculosis, COPD
7. X-ray chest abnormalities
8. ECG abnormalities

The patients and controls were evaluated as per the predesigned proforma and routine relevant investigations were done to assess their cardiac status and pulmonary status.

The routine scheme of evaluation included

1) History
2) General examination
3) Systemic examination
4) ECG

5) X-ray chest P/A view

6) Basic haemogram

7) Blood sugar, urea and serum creatinine

8) Echocardiogram

After assessing the baseline clinical and laboratory parameters patients and controls were subjected to computerized Spiro metric evaluation in batches after obtaining consent.

All the reading were expressed as the percentage of the predicted value for that particular Age, sex, height and weight comparable to south Indian population defined by Knudsen et al. Every test was repeated on three different occasions and the best of the three reading were taken.

The various spirometric parameters recorded were

1. Forced vital capacity (FVC)
2. Forced expiratory volume in the 1st Second (FEV_1)
3. Percentage of FVC expelled as FEV_1.

$$\frac{FEV1}{FVC} \times 100$$

4. Peak expiratory flow rate (PEFR)
5. Forced expiratory Flow rate at 50%
6. Forced expiratory Flow rate at 75%

Interpretation

The Spirometric readings are useful in the interpretation of the pattern of pulmonary dysfunction. The various abnormalities are

I. Restrictive pattern

Establish restriction – FVC < 80%

II. Obstructive Pattern

Established obstruction – $FEV_1/FVC < 75$

Early large airway obstruction – PEFR < 70% and $FEV_1 < 80\%$

Early small airway obstruction – $FEF_{50} < 70\%$ $FEV_{75} < 70\%$

II. Mixed Patter

1. When both FEF/FVC < 75% and FVC <80%
2. When FEFR < 70%, FEF_{75}<70% and FVC < 80%

RESULTS AND STATISTICAL ANALYSIS

A. COMPARISON OF PARAMETERS IN STUDY CASES AND CONTROLS

Table 1

Age

Age Group	Study Cases		Controls	
	No.	%	No.	%
< 20	6	10	2	10
20-29	22	36.7	7	35
30-39	20	33.3	7	35
40-49	10	16.7	3	15
50 & above	2	3.3	1	5
Total	60	100	20	100
Mean	31.4		32.3	
S.D.	9.2		10.8	
'p'	0.9734			

The mean age of study cases is 31.4 and the mean age of controls is 32.3. There is no statistically significant difference in the mean age of the study cases and controls.

Table 2 Sex

Sex	Study Cases		Controls	
	No.	**%**	**No.**	**%**
Male	36	60	10	50
Female	24	40	10	50
'p'	0.6014			

In this study, total number of study cases were 60. Out of 60, males were 36 (60%), Females were 24 (40%). Total number of controls were 20, out of 20, males were 10 (50%) females were 10 (50%). The Sex composition of the two groups does not differ significantly.

Table 3

Parameters	Study Cases		Controls		p
	Mean	S.D.	Mean	S.D.	
EVC	40.55	13.17	65.6	9.75	**0.0001(Significant)**
FEV_1	57.98	14.5	99.4	12.48	**0.0001(Significant)**
FVC	51.96	13.89	87.05	8.2	**0.0001(Significant)**
PEF	46.97	21.2	74.2	16.23	**0.0001(Significant)**
FEV_1/FVC%	114.17	6.85	119.2	4.13	**0.0073(Significant)**
MEF_{50}	60.23	28.58	92.4	8.23	**0.0001(Significant)**
MEF_{75}	48.28	26.97	75.25	12.95	**0.0001(Significant)**

There is statistically significant difference in the pulmonary function values between the Study cases and normal cases.

TABLE 4

INTERPRETATIONS OF CASES IN THE TWO GROUPS

Interpretations	Study Cases		Controls	
	No.	%	No.	%
Normal	2	3.3	20	100
Mild Restriction	19	31.7	-	-
Moderate Restriction	15	25	-	-
Severe Restriction	24	40	-	-

'p' = 0.0001 (Significant)

In this study, total number of patient were 60, out of 60, 2 patients did not have any pulmonary dysfunction. Mild restriction was seen in 31.7% of patients. Moderate restriction was seen in 25% of patients. Severe restriction was seen in 40% of patients. No pulmonary dysfunction was seen in controls (20 in No)

B. RELATIONSHIP OF VARIOUS PARAMETERS WITH INTERPRETATION IN THE STUDY CASES

Table 5

Age and Interpretation

Age	Interpretation							
	Normal		Mild Restriction		Moderate Restriction		Severe Restriction	
	No.	%	No.	%	No.	%	No.	%
≤ 20 (6)	-	-	3	50	1	16.7	2	33.3
21-30 (22)	1	4.5	7	31.8	6	27.3	8	36.4
31-40 (20)	-	-	6	30	6	30	8	40
41-50 (10)	1	10	2	20	2	20	5	50
> 50 (2)	-	-	1	50	-	-	1	50
Total (60)	2	3.3	19	31.7	15	25	24	40
Mean age	38		30.7		29.8		32.4	
S.D.	12.7		9.5		8.4		9.4	
	0.7298 (not significant)							

There is no statistically significant difference in pulmonary function test with respect to age.

TABLE 6

SEX AND INTERPRETATION

	Interpretation

Sex	Normal		Mild Restriction		Moderate Restriction		Severe Restriction	
	No.	%	No.	%	No.	%	No.	%
Male (36)	1	2.8	10	27.8	7	19.4	18	50
Female (24)	1	4.2	9	37.5	8	33.3	6	25
P	0.5434 (not significant)							

The total number of study cases were 60 out of 60, 36 patients were Males, 24 patients were female. Male patients out of 36 female patients, 1 patient was (2.8%) found to have normal pulmonary functions. 10 patients (27.8%) were found to have mild restriction, 7 patients (19.4%) patients were found to have moderate restriction. 18 patients were (50%) were found to have severe restriction. There is no statistically significant difference in pulmonary functions owing to gender difference.

Female patients.

Out of 24 Female patients, 1 patients (4.2%) was found to have normal pulmonary function tests. 9 patients (37.5%) were found to have mild restriction. 8 patients (33.3%) were found to have moderate restriction. 6 patients (25%) were found to have severe restriction.

TABLE 7

NYHA AND INTERPRETATION

NYHA Class	Interpretation							
	Normal		Mild Restriction		Moderate Restriction		Severe Restriction	
	No.	%	No.	%	No.	%	No.	%
Class I (9)	1	11.1	6	66.7	2	22.2	-	-
Class II (26)	1	3.8	13	50	11	42.3	1	3.8
Class III (25)	-	-	-	-	2	8	23	92
P	**0.0064 (Significant)**							

NEWYORK HEART ASSOCIATION CLASSIFICATION

FOR DYSPNOEA.

Class I No symptoms with ordinary physical activity.

Class II Symptoms with ordinary activity, slight limitation of physical activity.

Class III Symptoms with less than ordinary activity, marked limitation of activity.

Class IV Symptoms with any physical activity or even at rest.

Out of 60 study cases, 9 patients were with Class I dyspnoea, 26 patient were with Class II dyspnoea, 25 patients were with Class III

dyspnoea. Class IV dyspnoea patients were not included in this study because of they were haemodynamically unstable.

In this study, Class III dyspnoea patients were (25%) found to have severe restrictions (92%)

Table 7A: NYHA and MVO size

NYHA Class	MVO Size		
	Range	**Mean**	**S.D**
I	0.8 – 1.2	0.96	0.16
II	0.6 – 1.2	0.87	0.16
III	0.52 – 0.9	0.7	0.11
'p'	**0.0001 (Significant)**		

Statistically significant relationship exists between NYHA Class and MVO size.

Table 7B: NYHA Class and mean MV Gradient.

NYHA Class	Mean MV Gradient		
	Range	**Mean**	**S.D**
I	8 – 23	13.78	5.45
II	8 – 34	16.92	6.28
III	10 – 32	21.04	6.48
'p'	**0.0079 (Significant)**		

As NYHA Class increases, mean MV gradient values also increase. This relationship is statistically significant.

Table 8

Size of MVO (Cm^2) and Interpretation

Interpretation	Size of MVO (Cm^2)		
	Range	**Mean**	**S.D.**
Mild Restriction	0.8-1.2	0.98	0.15
Moderate Restriction	0.7-1	0.81	0.1
Severe Restriction	0.52-0.9	0.69	0.11
p	**0.0001 (significant)**		

As degree of restriction becomes Severe Restriction, mean MVO values decrease. This decrease is statistical significant.

Correlation between MVO size and mean MV gradient (r) = 0.53 r > 0.5

MVO size and mean MV gradiant are correlated.

Table 9

Mean MV Pressure gradient (mm Hg) and Interpretation

Interpretation	Mean MV Pressure gradient (mm Hg)		
	Range	**Mean**	**S.D.**
Mild Restriction	8-23	13.6	4.5
Moderate Restriction	10-28	18.7	5.8
Severe Restriction	10-32	21.2	6.1
p	**0.0002 (Significant)**		

As the mitral valve pressure gradient increases the degree of restriction become severe.

Table 10

LA CLOT AND INTERPRETATION

LA Clot	Interpretation							
	Normal		Mild Restriction		Moderate Restriction		Severe Restriction	
	No.	%	No.	%	No.	%	No.	%
Present (6)	-	-	1	16.7	-	-	5	83.3
Absent(54)	2	3.7	18	33.3	15	27.8	19	35.2
P	0.3067(Not significant)							

The patients with left atrial clot found to have Severe restrictive pulmonary functions (83.3%).

Table 11

EVC % and Interpretation

Interpretation	EVC %		
	Range	Mean	S.D.
Mild Restriction	25-71	47.4	13.2
Moderate Restriction	25-67	45.3	11.7
Severe Restriction	15-54	31.9	9.6
p	**0.0005 (Significant)**		

Table 12

FEV1 and Interpretation

Interpretation	FEV1		
	Range	**Mean**	**S.D.**
Normal	80-90	85	7.1
Mild Restriction	61-74	68.7	4.3
Moderate Restriction	46-69	62.5	7.5
Severe Restriction	24-70	44.4	11.1
p	**0.0001 (Significant)**		

Table 13

FVC and Interpretation

Interpretation	FVC		
	Range	**Mean**	**S.D.**
Normal	72-81	76.5	6.4
Mild Restriction	60-75	64.1	4.5
Moderate Restriction	32-60	55	6.9
Severe Restriction	20-61	38.4	8.8
p	**0.0001 (Significant)**		

Table 14

PEF and Interpretation

Interpretation	PEF		
	Range	**Mean**	**S.D.**
Mild Restriction	20-103	54.4	25.1
Moderate Restriction	21-96	50.2	22.9
Severe Restriction	15-66	38.6	12.8
p	0.1539 (not significant)		

Table 17

FEV1/FVC and Interpretation

Interpretation	FEV1/FVC		
	Range	**Mean**	**S.D.**
Mild Restriction	99-120	111.5	7.5
Moderate Restriction	98-123	114.2	0.7
Severe Restriction	104-125	116.2	6.2
p	0.1053		

Table 18

MEF_{50} and Interpretation

Interpretation	MEF_{50}		
	Range	**Mean**	**S.D.**
Mild Restriction	4-135	64.2	35.8
Moderate Restriction	26-117	70.4	29.3
Severe Restriction	20-89	50.1	18.9
p	0.1503(not significant)		

Table 19

MEF_{75} and Interpretation

Interpretation	MEF_{75}		
	Range	**Mean**	**S.D.**
Mild Restriction	6-137	55.5	33
Moderate Restriction	23-105	55.3	28.1
Severe Restriction	6-90	39.2	19
p	0.2109(not significant)		

STATISTICAL ANALYSIS

This study included 60 Patients of Isolated Rheumatic mitral stenosis aged between 10 and 51 years and 20 patients of control between 18 and 59 years

Male patients were 36 in group I

Female patients were 24 in group I

In group II (Control), the Male Female ratio was 1:1

There was statistically significant difference in the pulmonary function values between the study cases and normal cases of the 60 patients in group I, 2 patient did not have any pulmonary dysfunction whereas 19 patients (31.7%) were found to have mild restrictive pattern in pulmonary function, 15 patient (25%) were found to have moderate restrictive pattern, and 24 patient 40% were found to have severe restriction.

In group II (Controls), all 20 patients were found to have no pulmonary dysfunction.

Spirometric parameters

Controls Vs cases

On studying and comparing the spirometric parameters of controls and cases, it was found to have statistically significant reductions of EVC, FEVI, FVC, PEF, FEV_1/FVC%, MEF_{50} and MEF_{75}.

96.7% of patients were found to have mild to severe form of restriction.

As NYHA class increases, the restriction also increase.

As mitral value size reduces, the restriction also increase.

As the mitral value gradient values increase, grading of dyspnoea (NYHA class) increases mitral valve size and mean MV gradient are correlated.

DISCUSSION

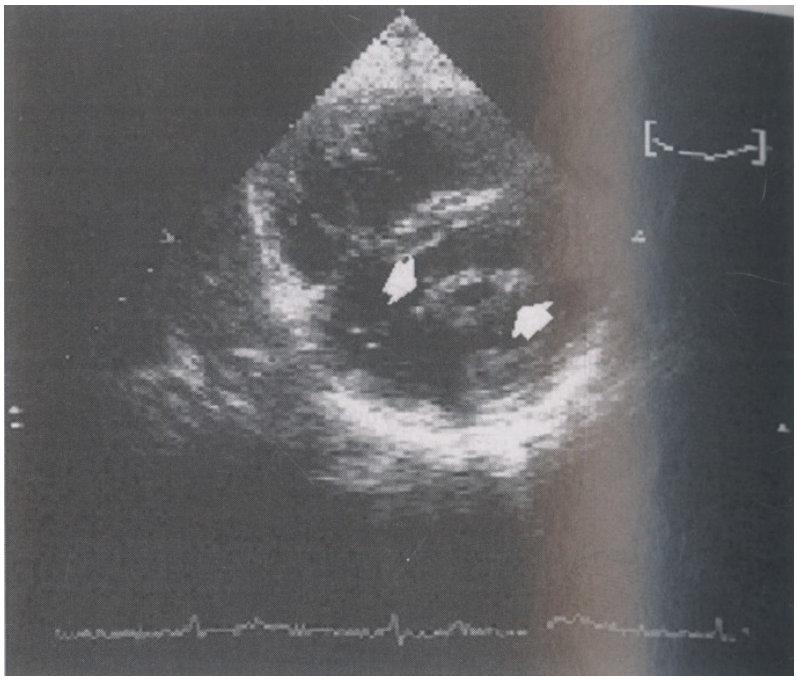
The incidence and prevalence of Rheumatic Fever world wide are enormous. The prevalence of Rheumatic Fever and RHD is highly variable, with the highest rates in the middle eastern and sub-saharan African regions. For instance, it is estimated that 3.2 million patients had rheumatic heart disease in India in 1991 and atleast two thirds of these were young children. Rheumatic Fever is a major cardiovascular health problem in many nations of the world.

Chronic valvular heart disease develops in at least half of those affected by rheumatic fever with carditis. Two-thirds of cases occur in women. Some episodes of rheumatic fever may pass unrecognized and it is only possible to elicit a history of rheumatic fever or chorea in about half of all patients with chronic rheumatic heart disease. The mitral valve is affected in more than 90% of cases; the aortic is the next most frequently affected valve, followed by tricuspid and then the pulmonary valve. Isolated mitral stenosis accounts for about 25% of all cases of rheumatic disease and an additional 40% have mixed mitral stenosis and regurgitation.

In normal adults the mitral valve orifice is 4-6 cm^2. In the presence of significant obstruction, (when the orifice is less than approximately 2cm^2), blood flow from the Left Atrium to the left ventricle (LV) only if

propelled by an abnormally elevated left atrio-ventricular pressure gradient. This is the hemodynamic hall mark of mitral stenosis. When the mitral valve opening is reduced to $1cm^2$, often referred to as "critical" MS, a Left Atrial pressure of approximately 25 mm Hg is required to maintain a normal cardiac output. The elevated pulmonary venous and pulmonary arterial wedge pressure reduce pulmonary compliance, contributing to exertional dyspnoea.

Apical 4 chamber view showing Fish-mouth appearance of severe rheumatic mitral



The Rheumatic mitral stenosis patients are more prone to develop pulmonary dysfunction and the severity of mitral stenosis may influence the degree of lung function impairment. The clinical and haemodynamic features of mitral stenosis are influenced importantly by the level of the pulmonary arterial pressure. Fibrous thickening of the walls of the alveoli and pulmonary capillaries occur commonly in mitral stenosis. Pulmonary function abnormalities, like vital capacity, total lung capacity, maximum breathing capacity, and oxygen uptake per unit of ventilation are reduced. Airway resistance is abnormally increased and diffusion capacity is also reduced. These changes in the lungs are due, to increased transudation of fluid from the pulmonary capillaries into the interstitial and alveolar spaces. However, the increased capacity of the pulmonary lymphatic system to drain excess fluid retards the development of alveolar edema.

In the present study, 60 Isolated Rheumatic mitral stenosis patients regularly attending the medical OPD and cardiology OPD for treatment were selected for analysis. To avoid confounding effects of variables like smoking, pulmonary tuberculosis, bronchial asthma and respiratory infections, 20 non-smoking age matched population was selected as control, after excluding the above mentioned factors. The mean age of the patient population was 31.4 years and that of control was 32.3 years.

The Patient population and controls were matched with respect on age and sex. Statistical analysis revealed that age and sex did not statistically influence the pulmonary function abnormalities. It was observed that as the valve orifice diminishes there is progressive restriction in the pulmonary functions. Also as the mitral valve pressure gradient rises the pulmonary functions deteriorate producing progressive dyspnoea.

In a study of 105 patients with rheumatic mitral stenosis Hryniewiecki T, et al., demonstrated that 93.5% patients had lung function impairment predominantly restrictive pattern. They observed that as the severity of mitral stenosis increases, there is a progressive deteriation in the pulmonary function. In our study also, as depicted in Fig.7 there was a linear correlation between the severity of mitral stenosis and progressive restrictive pattern of pulmonary function abnormalities. It was documented in our study that 96.7% of patients with isolated rheumatic stenosis had features of restrictive pattern of pulmonary abnormality.

The spirometric evaluation of the patients revealed features of restrictive pattern of pulmonary function abnormality in 96.7% of isolated Rheumatic mitral stenosis patient. Mild restriction was seen in 31.7% of patients. Moderate restriction was seen in 25% of patients. Severe restriction was seen in 40% of patients.

The restrictive dysfunction observed in this study was consistent with the study done by Chattarji et al. They found that a moderate restriction defect and small airway defect which is found in cases of mitral stenosis, directly correlates to the pulmonary artery pressure, left atrial pressure, mitral valve area and transmitral gradient. There was no significant involvement of the large airways. In our study also as demonstrated in Fig.6 there was an inverse correlation between mitral valve orifice area and severity of restrictive pattern of pulmonary function abnormalities.

In our study patient's symptoms according to NYHA classification correlated with the progressive decline of pulmonary function, mainly restrictive. In our study larger airways obstruction manifesting as an obstructive pattern of pulmonary function of abnormality was not observed. This again is consistent with the previous studies which stated that it is the smaller airway disease that dominates in mitral stenosis.

LIMITATIONS

1. The study was restricted to the hospital patients. So if relevance in general population is unknown.
2. Though carefully designed and meticulously carried out, the study is subject to subject error, instrument error and investigator error.
3. Class IV dyspnoea patients not included in this study.
4. Since the study was a cross sectional analysis, follow up the patients with treatment and potential reversibility with usage of bronchodilators as quoted by Nour MM, et al. could not be assessed.

CONCLUSION

Our study leads to the following conclusions

1. Most of the Isolated rheumatic mitral stenosis patients have mild to severe restriction of pulmonary function.
2. The Rheumatic mitral stenosis patients when compared with controls, showed statistically significant reduction in EVC, FEVI, FVC, PEF, FEVI/FVC%, MEF_{50} MEF_{75}.
3. The degree of restriction is correlated with grade of dyspnoea (NYHA Class).
4. As the mitral valvular size decreases, the grading of dyspnoea (NYHA Class) increases.
5. As the mitral valve gradient increases, the degree of restriction also increases.
6. As the mitral valve size decreases, the mean mitral valve gradient increases.
7. As valvular size decreases, the degree of restriction increases.

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PULMONARY FUNCTION TESTS IN ISOLATED

RHEUMATIC MITRAL STENOSIS

1. Name 2. Age 3. Sex

4. H/o. Hypertension, IHD, Aortic Valve disease, or any other congenital heart disease, bronchial asthma, pulmonary tuberculosis, COPD, H/o. smoking.

5. Any Respiratory disease :

6. Any Specific Complaints :

7. Clinical Features :

a. PR b) BP c) RR d) Chest expansion e) Temp

f) JVP g) Clubbing h) Dyspnoea NYHA class I) CVS

J) RS k) Abdomen l) Nervous System

8. Investigations

a) RBS b) Urine — Alb, Sugar, Deposits c) Blood — TC, DC, Hb

d) Blood Urea e) Serum Creatinine f) ECG

g) X Ray – Chest h) ECHO i) Others

LVIDd (cm) LIVDs (cm) LVEF %

RA, RV, LA & LV

Both the leaflets of MV

Thickness		MV Score for MS	
Subvalvular fusion		Mobility Restriction	
LA Clot		Calcification	
MVO	(sq.cm)	Pulmonary hypertension	

Doppler studies Peak gradient Mean gradient

IMPRESSIONS

J. Pulmonary Function Test

Para Meter	FVC	FEV1	PEF	FEF50	FEF75	FEV1/FVC	EVC	Impression
% of Predicted								

14. Treatment Particulars

15. Complications

16. Conclusion

Fig. 1. COMPARISON OF AGE BETWEEN CASES AND CONTROLS

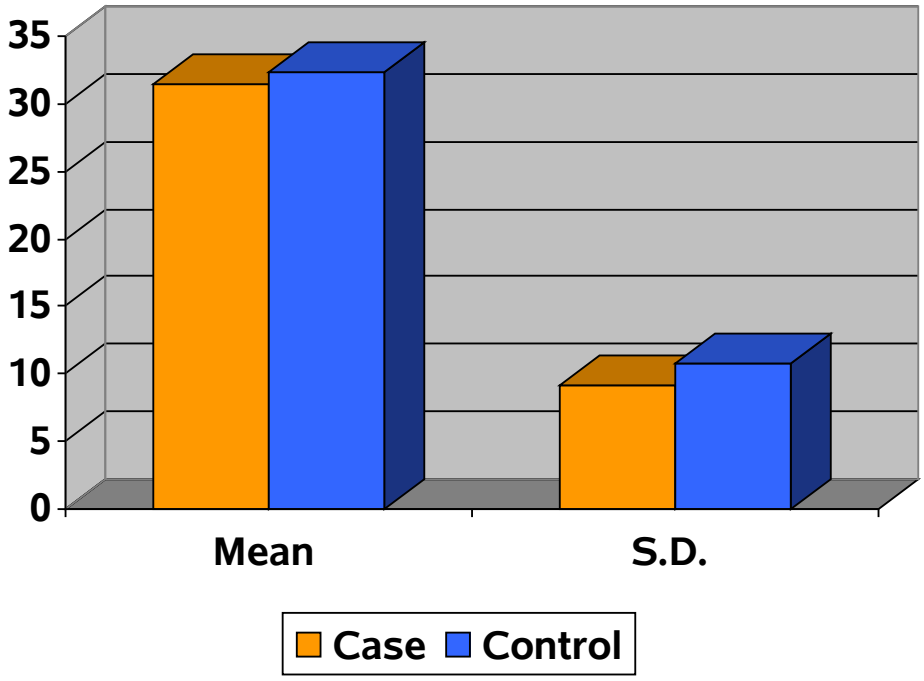

Fig. 2 SEX COMPOSITION OF THE STUDY POPULATION

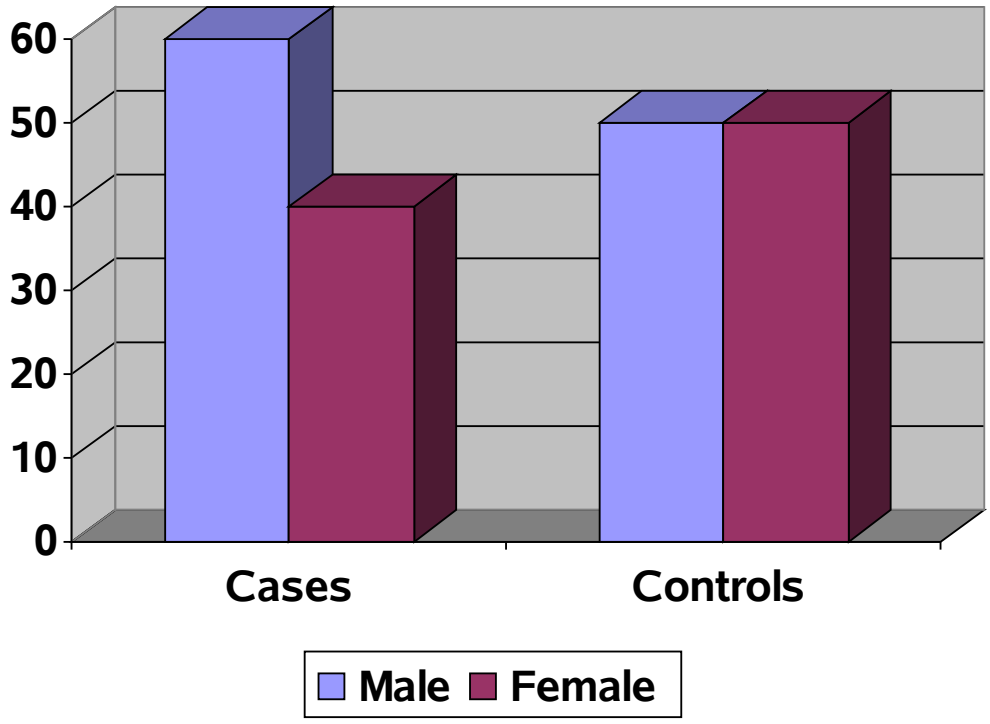

Fig. 3 COMPARISON OF PULMONARY FUNCTION INDICES BETWEEN CASES AND CONTROLS

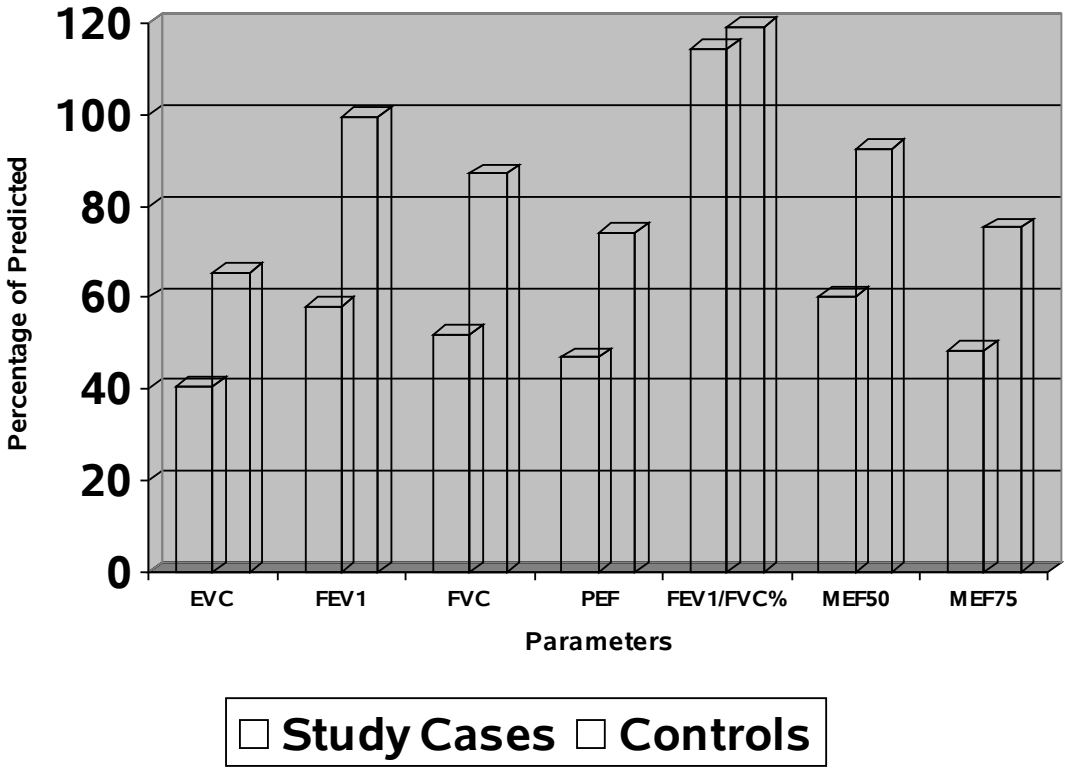

Fig. 4. PIE CHART SHOWING DISTRIBUTION OF ABNORMALITIES IN PULMONARY FUNCTION TEST IN CASES AND CONTROLS

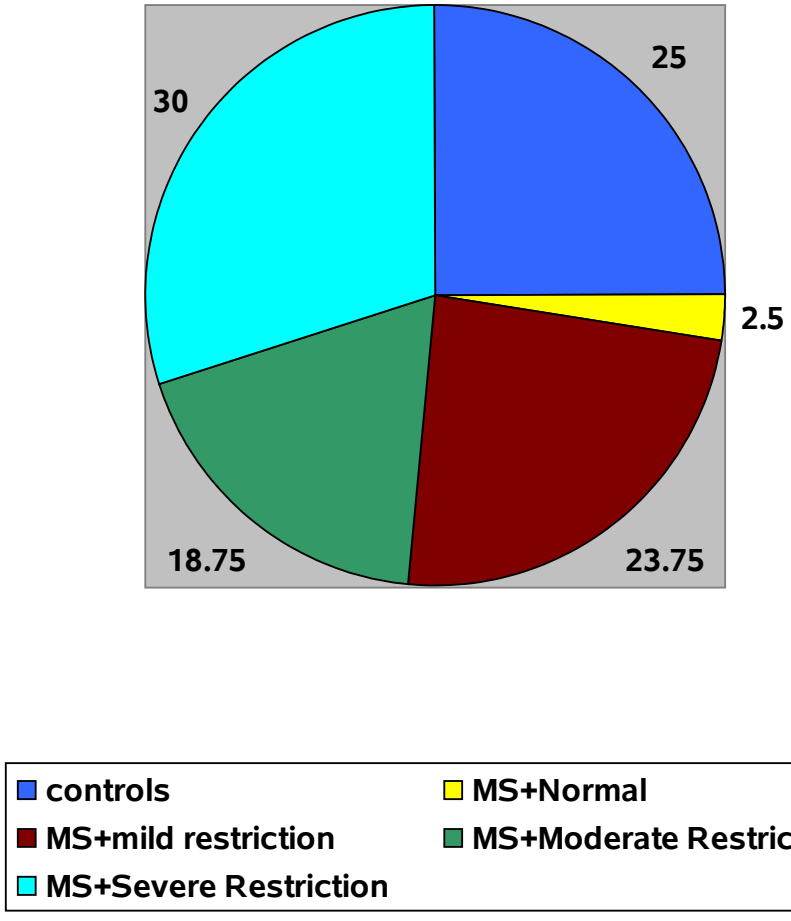

Fig. 5 CORRELATION OF SEVERITY OF SYMPTOMS AND ABNORMALITIES IN PULMONARY FUNCTION TEST

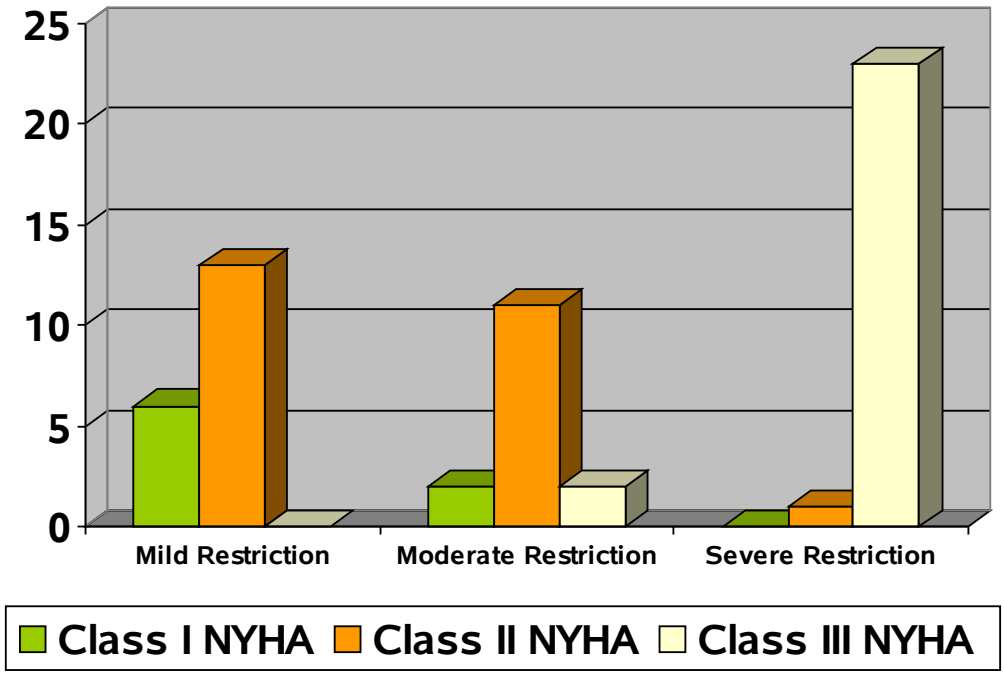

Fig. 6. CORRELATION BETWEEN SIZE OF MITRAL VALVE ORIFICE AND PULMONARY FUNCTION

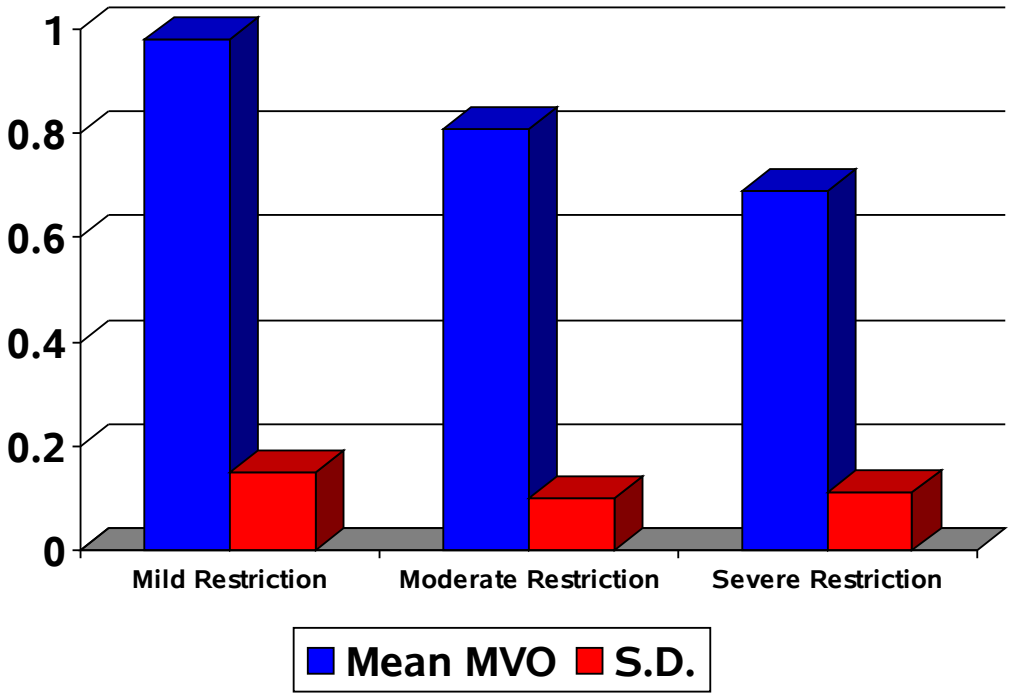

Fig. 7. CORRELATION OF MEAN MITRAL VALVE PRESSURE GRADIENT (MVPG) AND PULMONARY FUNCTION TEST